DOI: https://doi.org/10.53350/pjmhs22161434

ORIGINAL ARTICLE

Evaluation of Variations of Qt Interval in Hypothyroidism Through Autonomic and Vascular Profiling of Cardiac Function

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ABSTRACT

Background Hypothyroidism increases the risk of cardiac disease as well as autonomic dysfunction (abnormalities of the autonomic nervous system). A prospective case-control study was conducted. The purpose of this study was to compare the QT variability and vascular stiffness of SH patients.

Materials and Methods:Using ABI, we compared female SH patients to age- and sex-matched normal controls in order to better understand their condition. HF (0.15–0.4 Hz) and LF (0.003–0.04 Hz) frequencies were investigated spectroscopically. The HF represents vagal regulation, whereas the LF reflects both vagal and limbic regulation.

Result In this case research 32 women participated who were 28.02+7.8 years old and 28 women in control group who were age-matched (30.2+ 7.1). Patients had greater QT variability indices (P = 0.01) than healthy controls. Cases showed a higher LF/HF ratio than controls (P = 0.03), which was statistically significant. It was shown that there was a 0.02 point difference in left-right ABI between patients and controls.

Conclusion In these circumstances, sympathetic activity predominated over parasympathetic activity, with less of the latter. QTvi may be beneficial in determining the risk of cardiovascular mortality in this particular patient population.

INTRODUCTION

When there is no clinical hypothyroidism present, subclinical hypothyroidism is defined as having elevated thyroid stimulating hormone (TSH) in the presence of normal free thyroid hormone levels. Thyroid stimulating hormone (TSH) levels grow in patients with this condition, increasing their risk of developing overt thyroid failure. Patients with SH may have increased TSH levels that are not due to pituitary compensation for hyperthyroidism, but rather due to mild tissue hypothyroidism, which would explain the elevated levels of TSH. In addition to having high levels of atherogenic lipids and hypercoagulability, modest cardiac abnormalities (mostly diastolic dysfunction) and vascular dysfunction are all risk factors for developing SH. A modest thyroid hormone deficiency, as seen here, might have a deleterious influence on the cardiovascular system, as well. SVF (left ventricular dysfunction) and DVF (right ventricular dysfunction) are two kinds of heart failure that are distinct from one another (RV). It has been demonstrated that normalizing one's lipid profile can lessen one's cardiovascular risk ratio. Mild TSH elevations are handled if the patient's thyroid function test results are within normal limits. However, levothyroxine increases the chance of developing cardiovascular disease as indicated by carotid intima medium thickness and the formation of carotid artery plaque. An unintended consequence of primary hypothyroidism is autonomic dysfunction. The hormone estrogen alters the lipoprotein profiles of premenopausal women, increasing their risk of developing heart disease.

MATERIALS AND METHODS

With informed consent, we enrolled 32 drug-naive SH women (20–45 years old). We matched age and sex of controls. BMI >30 kg/m2, steroids, high dose beta blockers, lithium, major depression or severe anxiety disorder were all excluded from the experiment. Major medical diseases like pulmonary, cardiac, hepatic, renal, and neurological illnesses were excluded. Diabetic and non-diabetic patients having TSH 4.5-10 mU/L with normal freeT4 levels were included. Tests were performed in the morning, 3 hours after a light lunch and no caffeinated beverages. Patients were checked in the same lab after 10–15 minutes. The tests were noninvasive and performed while the patient slept. Here are the results. The study's outcome measures are listed below.

In eight leads, Delmar Digicorders recorded ECG signals at 1000 Hz with 1 ms accuracy (California, USA). These files were backed up on hard discs and CDs.

HRV can be expressed in time or frequency. An important advance is the introduction of spectral analysis to cardiac time series. Frequency-specific oscillations can be studied using spectral analysis. In this way, not only the variability but also the oscillation frequency may be determined. That which occurs between 0.15 and 0.5 Hz is the parasympathetic nervous system (high frequency [HF]). They are both controlled in this spectral peak. These devices appear to regulate VLF and ultra LF spectral power. The LF/HF power ratio is a useful but controversial derived measure of sympathovagal balance. This respiratory sinus arrhythmia is promoted by recumbent position and controlled breathing.

The "R" wave timing was obtained using a graphical interface of digitized ECG. A QT wave template's start and end were entered into the program. The time stretch model calculates each beat's QT interval. Choosing a longer QT template shifts all QT intervals. Not the mean QT, but the QT variability. The RR and QT intervals were collected at 4 Hz to provide instantaneous HR and QT. A 4 Hz sampling rate estimates the power spectrum to 1 Hz. Software employed a linear spline to eliminate premature beats from these data. The data were detrended using the best fit line before spectral analysis. Each limb had oscillometric BP cuffs. A PCG sensor was placed on the V2 point on the chest. Inflate and deflate all four cuffs while monitoring lead I ECG and PCG. The right carotid artery had a collar-shaped carotid sensor. We created two 30s records. The technology calculated all vascular indicators automatically. The samples were 1024 Hz. The Vascular Profiler provided the following metrics: SBP, DBP, MAP, and pulse pressure are 60-second averages. MAP = 1/3 DBP (SBPDBP). The PWV is the speed at which the heart's pulse reaches the end artery mainly to measure artery wall hardness. PWV + L/pulse Axilla to tibi PWV Statistics were used to calculate the distance. Stiffened arteries Normal population values and gender influenced arterial stiffness percent. It used larger brachial SBP. Pseudosynovitis is an atherosclerosis test. A score of 0.9 or below often indicates peripheral occlusive vascular disease in the lower limbs. A vascular stenosis score of 1.2 or 1.3

The device recognized the heart contraction's arterial pulse as cuff pressure oscillation. We calculated BP using the relationship between cuff pressure and oscillation. Fast cuff pressure oscillations were measured as SBP and DBP. The MAP was taken during the oscillation's peak. There was no influence from external noise. An eight-lead ECG was acquired in a quiet environment. An artifact-free 256 s data set captured at 1024 Hz (QTvi).

RESULTS

Outpatients in the field of endocrinology one physiologist examined the function of the autonomic nervous system. It is not known if SH sufferers have sympathetic or parasympathetic nervous systems. A total of 32 female SH patients were compared to 28 euthyroid controls who were age, sex, and BMI matched to the SH patients. It was discovered that the HRLFHF of patients was greater than that of controls (P=0.02). Cases differed much more from controls.

Variables	Cases	Controls	*P*
	Mean±SD	Mean±SD	
	Group 1 (*n*=32)	Group 2 (*n*=28)	
Age (years)	28.02+7.8	30.2+ 7.1	0.87
TSH (mIU/ml)	5.77±1.27	2.33±0.81	0.01
BMI (kg/m²)	25.56±5.48	26.14±4.72	0.05
HRR	801.12±111.14	807.13±111.01	0.87
HRLFHF	1.42±1.03	0.88±0.52	0.01
QTRT	410.02±30.01	389.01±55.12	0.68
RRHF (LN)	6.401±1.14	6.601±1.02	0.07
QTvi	0.866±0.501	0.822±0.601	0.01
SBP right (mmHg)	112.78±17.82	112.00±11.82	0.27
DBP right (mmHg)	65.36±7.12	65.62±7.88	0.69
SBP left (mmHg)	110.88±18.72	114.3±10.43	0.32
DBP left (mmHg)	66.42±7.98	67.70±8.11	0.31
Left ABI to minus right ABI	0.423±0.27	0.419±0.30	0.02

Table 1: Parameters in cases and controls

The RRHF log indicated that there was a control bias (P = 0.08). The RRHF of controls was greater than that of patients. SBP, DBP, and PWV were all comparable across cases and controls in both cases and controls. The TSH/LF/HF ratio did not have a significant correlation (P = 0.73, r = 0.03).

DISCUSSION

During our research, we discovered that the SH patients had atherosclerosis. In this study, only girls with similar age, BMI, and gender were employed, and no men were included.

The activity of the parasympathetic nervous system is reduced in SH. HRHFLF was greater in patients (P = 0.02) than in controls. This is consistent with the findings of Galetta et al. They were both male and female, while Galleta's study included more females than males, as well as patients over the age of 65. They were 31.8 plus 8.9 years old at the time. Autonomic impairment has been observed in hypothyroidism by Mahajan et al. According to Inukai et al., hypothyroidism increases sympathetic cardiac input. Power spectrum analysis is more sensitive than other methods. It was revealed that patients had lower heart rates than controls, albeit not by a significant amount. According to HRV, SH sufferers have a parasympathetic nervous system that is not functioning properly. QTvi, an indicator of QT variability adjusted for mean QT over HRV, revealed that there was a sympathetic preponderance in patients (P = 0.01), as judged by the patients. This delay between cardiac depolarization and repolarization is referred to as the QT interval. If you have SH, you may notice that your ventricular recovery time is not uniform. In a recent study, Atiga and colleagues revealed that QTvi is a more accurate predictor of sudden cardiac mortality than ejection fraction, heart rate variability, and T wave alterans. Despite the fact that QT fluctuation often accompanies HRV, there may be some incoherence in the data. Adult coherence between variations in the heart rate and the QT interval was found to be lower than that found in children in the high-frequency range (0.15–0.5 Hz). QTvi is raised by intravenous isoproterenol and rising from a supine position, both of which increase sympathetic activity. It has been demonstrated that both palmolein and yohimbine, which are sympathomimetic drugs, raise QTvi, showing that they have a sympathetic link. Consequently, greater QTvi in SH patients indicates increased sympathetic activity and, as a result, increased cardiovascular morbidity. Anecdotal evidence suggests that hypothyroidism is accompanied by sympathetic over activity. Hypothyroidism, on the other hand, was revealed by Xing et al. to be associated with increased vagal tone. Inconsistencies in results may be explained by differences in patient selection, monitoring techniques, and the type, severity, and duration of hypothyroidism seen in different studies. Controls were preferred above cases according to the RRHF record. According to Galetta et al., high TSH levels were associated with QT dispersion and returned to normal once medication was administered. In hypothyroid individuals, Kahaly found a dysfunction of the parasympathetic nervous system. The participants in this study were older women who were tested for their heart rate response to exercise and recovery. HrV was found to be decreased in these

situations. Our research showed no evidence that lower HRV increases the chance of developing an arrhythmia (P = NS). The sympathetic tone of the patient increases as the heart rate decreases, indicating a decrease in vagal tone. In our study, patients' heart rate variability (HRV) was lower than that of controls. Sahin et al. discovered that there was a decrease in sympathetic tone and an increase in parasympathetic activity. Hypothyroidism was revealed by Inukai et al. to be caused by a parasympathetic deficit. Celik and colleagues discovered that levothyroxine did not restore cardiac autonomic function in the patient group. The researchers observed that patients with SH exhibited lower time domain HRV characteristics than the control group, with statistically significant variations in SDNN, RMSD, triangular interpolation of NN interval, and mean response ratio compared to the control group. HF is a signal marker produced by vagal efferent modulation. Cases had lower heart rate variability (HRV) in both the temporal and frequency domains. The heart rate variability (HRV) measures autonomic cardiac input. The effects of autonomic withdrawal and sympathetic saturating input on heart rate variability (HRV) are investigated. Autonomic dysfunction is thought to be caused by hypothyroidism, which is thought to be caused by an increase in thyrotrophic releasing hormone and a decrease in chronotropic responsiveness to beta adrenergic stimulation. They can also increase extracellular protein deposition, resulting in water buildup in the heart wall, fibrosis, and inhomogeneity of ventricular repolarization dis different regions of the heart wall. The researchers discovered a statistically significant relationship between TSH and the root mean square of successive RR intervals in people with TSH greater than 10.Its impact on sympathetic and parasympathetic nervous system activity is unknown at this time. Our findings were similar to those of Galetta et al. in that they showed increased sympathetic activity and decreased parasympathetic activity. Neither autonomic withdrawal nor excessive sympathetic activity have been shown to enhance heart rate variability (HRV). SH and hypothyroidism, according to Hamano and Inoue, cause increased vascular wall stiffness, which can be reversed with effective therapy of the condition. SH, according to Kosar F et al., is associated with decreased right ventricular diastolic performance. The PWV of the patients and the controls were the exact same. Sympathetic or parasympathetic dysfunction may be present in SH, which should be considered while treating the condition. SBP and DBP were comparable in their results. There were no cases of hypertension in the subclinical hypothyroid group. The difference in ABI between the left and right sides was greater in instances (P = 0.03). A 1 mmHg difference between the left and right brachial systolic pressures, according to Nead and colleagues, increased the chance of all-cause mortality by 11 percent. [46] In this study, we found a variation in ABI between the right and left sides, which may be a predictor of cardiovascular morbidity in future investigations. Our research is limited by a lack of follow-up data with repeat autonomic function tests after taking levothyroxine, which we did not have. Following treatment, it is necessary to examine the measures taken by these patients in further detail. Because our study only involved females, more research is needed to determine whether similar advantages are also available to men.

CONCLUSIONS

While earlier research has mostly focused on vagal dysfunction, our data indicate that sympathetic dysfunction is also frequent in this group of individuals. Using the QTvi, it may be possible to estimate cardiovascular mortality in this patient cohort. In this study, the authors corroborate prior results that untreated SH may raise the risk of cardiovascular death. These findings show that individuals with SH who have a higher risk of cardiovascular morbidity and mortality should get replacement medication. It may pave the way for further research into the changes that occur in the vascular system throughout time**.**

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